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ANTIFUNGAL RESISTANCE PATTERNS OF CANDIDA SPECIES IN VULVOVAGINAL CANDIDIASIS: A FOCUS ON THE EMERGENCE OF NON *Albicans candida* AND THERAPEUTIC IMPLICATIONS

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ABSTRACT

Non-albicans Candida (NAC) species and antifungal resistance are becoming increasingly linked to vulvovaginal candidiasis (VVC), a frequent gynecological infection. The prevalence, species distribution, and antifungal susceptibility of Candida isolates in women of reproductive age were examined in this study. Fifty (50) subjects had their vaginal swabs taken, and they were then grown on Sabouraud Dextrose Agar and chromogenic agar. Biochemical responses and colony morphology were used to identify Candida isolates. Using established procedures, antifungal susceptibility to six widely used drugs was evaluated. Twenty-six (26) (52%) of the 50 subjects tested positive for Candida. The age group of 21–25 years old had the highest frequency (42.3%). The most isolated species was *Candida krusei* (34.6%), which was followed by *Candida albicans* (26.9%), *Candida tropicalis* (15.4%), *Candida parapsilosis* (15.4%), and *Candida glabrata* (7.7%). Antifungal tests showed high resistance to fluconazole (92.3%), clotrimazole (57.7%), and nystatin (53.8%), as well as universal resistance to amphotericin B. Miconazole, on the other hand, exhibited the highest activity (96.1% susceptibility). A notable change in the epidemiology of VVC is shown by the prevalence of fluconazole resistance and the preponderance of NAC species, particularly *C. krusei*. In this situation, miconazole continues to be the most effective antifungal medication. These findings emphasize the need for species-level identification; effective management depends on ongoing surveillance, antifungal care, and routine Candida speciation.

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INTRODUCTION

Vulvovaginal candidiasis (VVC) is an infection of the vaginal tract, often caused by *Candida albicans*, an opportunistic pathogenic yeast. It is the second most prevalent cause of vaginitis after bacterial vaginosis [1]. *Candida albicans* colonizes the gastrointestinal, reproductive, and epidermal systems, and can be present in 20-30% of healthy, asymptomatic women [2]. Non-albicans Candida species, such as *C. glabrata*, *C. parapsilosis*, and *C. tropicalis*, are also recognized as distinct causes of VVC [1,2].Recurrent vulvovaginal candidiasis (RVVC) affects 8-10% of women worldwide, with 158 million projected to be affected by 2030 [3,4]. Lifetime VVC rates range from 29 to 49%, with 9% reporting at least four episodes in a year [5]. The burden is particularly high for women aged 25-34 years. African environments have a higher prevalence of RVVC, suggesting local diagnostic and behavioral factors may impact the disease burden [4,6]. Non-albicans Candida species like *C. glabrata*, *C. krusei*, *C. parapsilosis*, and *C. tropicalis* are increasing in RVVC cases, accounting for up to 10-20 % [7,8]. These species often arise in recurrent or complex

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infections. Fluconazole-insensitive isolates nearly tripled between 2018 and 2021, with *C. albicans* accounting for 70% of isolates [9].

Antifungal resistance, particularly fluconazole, complicates RVVC treatment, diminishing conventional therapy effectiveness in NAC and *C. albicans* species. Reduced sensitivity to fluconazole, nystatin, and clotrimazole is linked to biofilm generation [10]. Resistant RVVC requires treatments like boric acid, nystatin, or intermittent azole maintenance regimens; boric acid is a first-line treatment with 72% cure rates [11]. Maintenance regimens include clotrimazole, miconazole, or boric acid; customized treatments based on species and response history are recommended. Recent systemic treatments include ibrexafungerp and oteseconazole, which have shown promising outcomes against azole-resistant Candida species [12].

Several factors contribute to the pathogenesis of RVVC, including host immune dysfunction, genetic predispositions (such as TLR2 or mannose-binding lectin polymorphisms), hormonal factors, diabetes mellitus, HIV, and behavioral factors like the use of antibiotics and vaginal hygiene practices. Douching, using feminine wash, or the effects of estrogen or antibiotics can all disrupt the balance of lactobacilli and encourage the growth of Candida, increasing the risk of recurrence [13].

Non-albicans Candida species, including *C. krusei*, *C. glabrata*, and *C. tropicalis*, are increasing in prevalence, particularly in low-resource environments like Nigeria. These species have developed resistance to common antifungal medications, leading to pregnancy-related complications. Diagnostic and therapeutic options are often based on outdated theories, causing financial strain, poor treatment, and high recurrence rates. Lack of up-to-date data on Candida species distribution and susceptibility profiles worsens patient suffering.

To improve diagnosis, provide appropriate medication, and guide public health policies for decreasing RVVC, it is essential to comprehend the local epidemiology and antifungal sensitivity patterns of Candida species. In Nsukka city, a population that is especially susceptible to RVVC, this study offers much-needed baseline data on the prevalence of Candida species and their antibiogram among women of reproductive age. In the end, this work establishes the foundation for evidence-based antifungal stewardship in the area and offers insightful information to the local medical community.

MATERIALS AND METHODS

Population Under Study

Fifty (50) women between the ages of sixteen and forty-five who had clinical symptoms suspected of vulvovaginal candidiasis (VVC) at the University of Nigeria Medical Centre and Bishop Shanahan Hospital in Nsukka, Enugu State, Nigeria, were included in this study. Six age groups (16–20, 21–25, 26–30, 31–35, 36–40, and 41–45 years) were used to stratify the participants. Women of reproductive age who were not menstruating at the time of sample collection and who had not taken antibiotics or antifungal medications in the two weeks prior met the inclusion criteria. To reduce confounding variables, women who were pregnant, going through menopause, or suffering from systemic diseases like diabetes mellitus were not included. The management of the participating medical facilities gave their approval for the study to be conducted. Confidentiality was guaranteed, and all participants received information about the study's goals, methods, and advantages. Before the sample was collected, each participant provided written informed consent. Participants were free to leave the study at any time without losing any benefits because participation was completely voluntary.

Preparation of Media

Sabouraud Dextrose Agar (SDA) (LabM, UK) Before being transferred into sterile petri dishes for gelling in a sterile setting, SDA was dissolved in distilled water, chloramphenicol combined with 70% ethanol was added, autoclaved, and cooled down [14].

Slant Culture: After preparing the SDA, about 7 ml of the prepared medium was poured into 30 bijou bottles and kept in a slant position to gel. After 24 hours of gelling, the fungal isolates were transferred to the slant culture and allowed to grow. The growth of the culture appeared after 48 hours.

Chromogenic Agar (Himedia, India) Thirteen grams of Hichrome chromogenic agar (Himedia India) were dissolved in distilled water, boiled, homogenized, and cooled before being poured into sterile petri dishes without autoclaving [15].

Gathering and Culture of Samples

Aseptic collection of high vaginal swabs (HVS) from the participants was carried out using sterile swab sticks with the help of a medical officer. It was inserted 2 inches into the vaginal opening and rotated against the vaginal wall for 15 sec and returned aseptically into the tube. All the samples were labeled appropriately and I took them to the laboratory for analysis. The samples were inoculated onto SDA and Chromogenic Agar . They were incubated at 35-37© for 24 to 48 hrs A McFarland turbidity standard (0.5) was prepared by adding 0.5 ml of 1.17% (W/V) Barium chloride solution to 99.5 ml of 1% (V/V) tetraoxosulphate(vi) acid, sealed, and stored at room temperature. The tube was shaken to mix the Barium sulphate precipitate.

Sensitivity Test

The Candida isolates were sub-cultured twice on SDA plates before antifungal susceptibility testing. An inoculating loop was used to transfer colonies into prepared SDA plates. All the candida isolates were subjected to Antifungal sensitivity test by disk diffusion method using Muelle -Hinton Agar (Oxoid, UK) + 2% glucose and 0.5 ug/ml methylene blue. Yeast suspension was adjusted to the McFarland standard , and was spread onto the prepareed plate. Antifungal agents were added to the plates, and incubated overnight. The drugs used include Amphotericin B (20 mcg), Clotrimazole (10 mcg), Nystatin (50 mcg), Fluconazole (25 µg), Miconazole (30 mcg), Voriconazole (1µg).

Reading of Plates and Measurement of Diameter of Zone of Inhibition

The plates were examined after 24 hours of incubation. The diameters of zones of inhibition were measured in millimeters using meter rule. The petri dish was viewed from the back against a dark background to increase contrast, the ruler was placed across the centre of the zone of inhibition and the full diameter of the clear zone was measured from one edge to the opposite edge and it was interpreted according to Clinical Laboratory Standard Institute (CLSI) breakpoint.

RESULTS

Of the 50 participants, 26 (52%) tested positive for Candida as shown in Table 1. Among women aged 21 to 25, the prevalence was highest (42.3%), followed by those aged 16 to 20 (26.9%). Among women aged 41 to 45, there were no positive cultures.

Table 1: Distribution of Vulvovaginal Candidiasis (VVC) by Age Group

Age	Positive for VVC	Negative for VVC	Total
16 – 20	7(26.9%)	8 (33.3%)	15 (30%)
21 – 25	11(42.3%)	4 (16.7%)	15 (30%)
26 – 30	5 (19.2%)	1 (4.2%)	6 (12%)
31 – 35	2 (7.7%)	3 (12.5%)	5 (10%)
36 – 40	1 (3.9%)	2 (8.3%)	3 (6%)
41 – 45	0	6 (25%)	6 (12%)
TOTAL	26 (100%)	24 (100%)	50 (100%)

According to Table 2, *Candida krusei* was the most prevalent isolate (34.6%), followed by *Candida albicans* (26.7%), *Candida tropicalis* (15.4%), *Candida parapsilosis* (15.4%), and *Candida glabrata* (7.7%).

Table 2: Biochemical Identification of Candida Species

Candida Sp.	Color on Chromogenic Agar	Number of Isolates
Candida krusei	Purple	9 (34.6%)
Candida albicans	Green	7(26.9%)
Candida parapsilosis	Pink	4 (15.4%)
Candida tropicalis	Blue	4 (15.4%)
Candida glabrata	White	2 (7.7%)

Table 3 shows that the majority of *C. krusei* isolates were detected in women aged 21–30, but *C. albicans* was more prevalent in women aged 21–35. It also demonstrates that among those aged 36 to 40, there was just one isolated case of *C. glabrata*.

Table 3: Distribution of Candida Species by Age Group

Age Group	*C. krusei*	*C. albicans*	*C. parapsilosis*	*C. tropicalis*	*C. glabrata*	Total (%)
16-20	2	1	3	1	-	7 (26.9)
21-25	3	3	1	3	1	11 (42.3)
26-30	4	1	-	-	-	5 (19.2)
31-35	-	2	-	-	-	2 (7.7)
35-40	-	-	-	-	1	1 (3.9)
41-45	-	-	-	-	-	0
Total (%)	9 (34.6)	7 (26.9)	4 (15.4)	4 (15.4)	2 (7.7)	26 (100)

Table 4 shows that Amphotericin B resistance was present in all isolates. High resistance was seen to voriconazole (69.2%), nystatin (53.8%), clotrimazole (57.7%), and fluconazole (92.3%). Conversely, miconazole was the most effective, rendering 96.1% of isolates susceptible.

Table 4: Antifungal Susceptibility of Candida Isolates (n = 26)

Anti-Fungal Agents	Susceptible	Intermediate	Resistant
Amphotericin B	-	-	26 (100%)
Clotrimazole	5 (19.2%)	6 (23.1%)	15 (57.7%)
Fluconazole	1 (3.85%)	1 (3.85%)	24 (92.3%)
Miconazole	25 (96.15%)	1 (3.85%)	-
Nystatin	5 (19.23%)	7 (26.92%)	14 (53.85%)
Voriconazole	3 (11.54%)	5 (19.23%)	18 (69.23%)

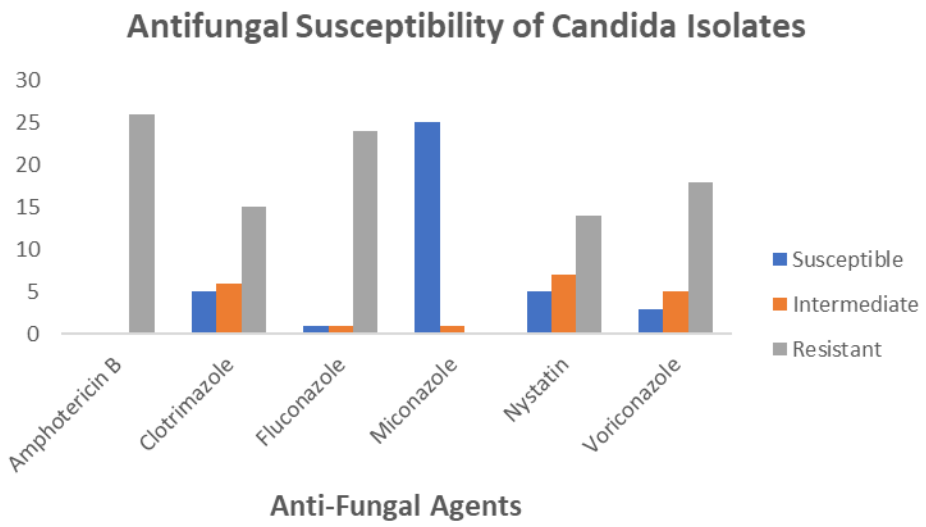

Figure 1: Antifungal Susceptibility of Candida Isolates

Figure 2: Chromogenic Differentiation of *Candida glabrata* (White) and *Candida tropicalis* (Blue)

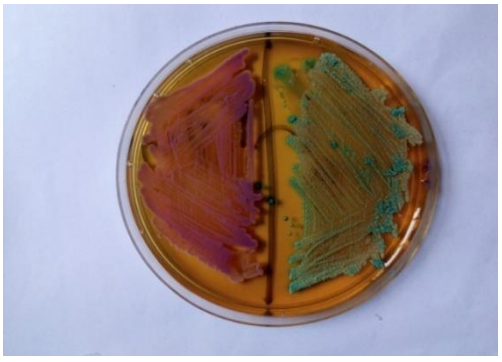

Figure 3: Chromogenic Differentiation of *Candida parapsilosis* (Pink) and *Candida albicans* (Green)

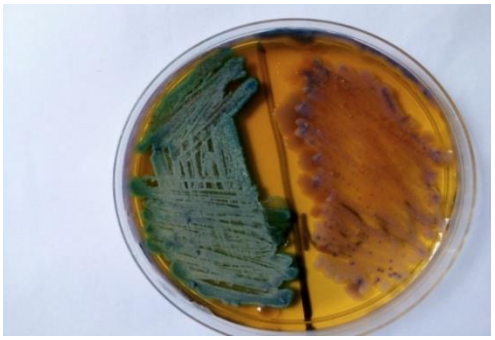

Figure 4: Chromogenic Differentiation of *Candida albicans* (Green) And *Candida krusei* (Purple)

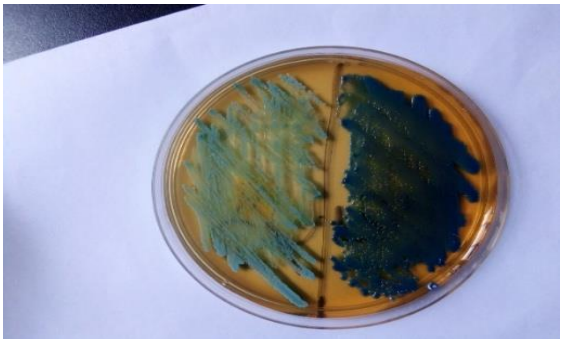

Figure 5: Chromogenic Differentiation of *Candida tropicalis* (Blue)

Figure 6: Zones of Inhibition (AP - Amphotericin B, CC - Clotrimazole, FCA - Fluconazole, MIC - Miconazole, NS - Nystatin, VOR – Voriconazole)

DISCUSSION

In this study, non-albicans Candida (NAC) species dominated the culture growth, with *C. krusei* (34.6%) leading, followed by *C. albicans* (26.7%), *C. parapsilosis* and *C. tropicalis* (15.4% each), and *C. glabrata* (7.7%). According to age-stratified counts, the load is highest among those aged 21 to 25 (42.3%), with significant contributions from those aged 16 to 20 (26.9%). The species mix changes with age: *C. parapsilosis/C. tropicalis* concentrated in younger groups (16–25 years), while *C. krusei* dominated in 26–30 years (4/5 isolates). These trends imply that NAC organisms are a significant cause of disease in this context and that the majority of culture-positive VVC were young, reproductive-age women. This pattern is consistent with epidemiological studies showing higher incidence of VVC in age groups exposed to estrogen and sexual activity, with the highest prevalence found in women aged 18-25 [5,16,17]. Estrogen in women promotes a glycogen-rich vaginal epithelium, lowering pH and fostering Lactobacillus dominance. If imbalanced by antibiotics or hormonal changes, Candida overgrowth may occur, raising yeast infections risk [18].

The prevalence of non-albicans Candida (NAC) in this study's species distribution is noteworthy; although NAC proportions are increasing, some modern cohorts still find *C. albicans* to be dominant (≈70–85%), with NAC making up 10–30% of the total [19]. This study found a rise in non-albicans yeasts which is consistent with a rise from 6.0% to 12.6% in a UK recurrent/complicated VVC cohort due to *C. glabrata* [8]. Although *C. glabrata* is usually the most common NAC in VVC, the top ranking of *C. krusei* (intrinsically fluconazole-resistant) in this study is not common but is clinically significant. It aligns with the larger worry that NAC shifts make azole-based regimens more difficult. According to recent reviews, *C. glabrata* and *C. krusei* are the main causes of azole treatment failure in RVVC [20,21].

With miconazole maintaining high activity, the results of this study show extremely high azole resistance (e.g., fluconazole resistance ≈92%). Miconazole and clotrimazole are two examples of topical imidazoles that frequently have good in vitro action against Candida spp. (94–99% susceptibility) [22,23,24], however, a research data shows rising rates of fluconazole non-susceptibility, especially in non-albicans species and recurrent *C. albicans* [25]. Fluconazole non-susceptibility rose from 3.5% to 9.6% in the Leeds RVVC cohort between 2018 and 2021 [8]. Recent clinic-based research suggests that miconazole, itraconazole, and amphotericin B have stronger action against vaginal isolates [17].

NAC (particularly *C. glabrata/C. krusei*) showed decreased azole susceptibility, highlighting the necessity for species-level identification and customized treatment. Azole resistance among NAC is on the rise, and more significantly, there is a discernible rise in azole-resistant *Candida albicans*, therefore, rather than being an isolated oddity, the high NAC share observed in this study especially *C. krusei* is consistent with new global susceptibility trends. [26,27]. A good comparable is the Leeds (UK) 2018–2021 RVVC cohort: over a three-year period, fluconazole-resistant isolates climbed from 3.5% to 9.6%, while NAC increased from 6.0% to 12.6% [8]. The Leeds study emphasizes the exact problem in this study's result: the RVVC group has higher levels of NAC and azole resistance, which is probably making empirical azole failure rates worse. This is highlighted by our antifungal susceptibility panel, which showed >50% resistance to clotrimazole and nystatin, 92.3% overall (24/26) fluconazole resistance, and 69.2% voriconazole resistance. In contrast, 96.2% of isolates maintained miconazole activity. The study suggests that fluconazole resistance in high NAC-content *C. krusei* could be a possibility, since *C. krusei* is inherently resistant to fluconazole, but topical imidazoles like miconazole can still maintain activity in VVC collections [17,28].

The consequences of this are that empirical fluconazole is unlikely to work in areas with high NAC incidence; instead, therapy should be guided by species-level identification and knowledge of local susceptibility; and, where NAC or azole resistance is suspected, this study suggests azole-sparing alternatives for induction/maintenance: topical miconazole appears to be highly efficacious locally. These decisions align with current breakpoints guidelines and recent reviews that focused on resistance [8,20].

CONCLUSION

This study demonstrates a shift in the epidemiology of VVC toward non-albicans Candida species, with *C. krusei* emerging as the most prevalent isolate. Miconazole's remarkable activity persisted, but there was a startlingly high level of resistance to commonly used antifungals, particularly fluconazole. These findings emphasize the need for species-level identification, careful antifungal usage, and continuous monitoring to optimize therapy outcomes.

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AUTHORS' CONTRIBUTION

CBC, COO and BOO were responsible for conceptualization and methodology; CBC, COO and FOO carried out the investigation and wrote the initial draft of the manuscript; CCA and NWE reviewed the draft, FOO did the statistical analysis. All authors reviewed and approved the final manuscript.

CONFLICT OF INTEREST

The authors declare no conflicting interests.

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